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Acute kidney injury and paralytic ileus- An unusual presentation of hypothyroidism

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Abstract- A 55 year old male patient, known case of hypothyroidism since last four years was admitted to our nephrology unit in a state of drowsiness and confusion. He had paralytic ileus and impaired renal functions at the time of admission. On routine investigations hypokalemia was present, which likely contributed to the paralytic ileus and together with dehydration had lead to renal injury. Nonetheless, hypothyroidism would have been the principal precipitant of both these complications. Both paralytic ileus and acute kidney injury improved with thyroxine replacement.

Hypothyroidism may induce de novo acute kidney injury or it may exacerbate ongoing chronic kidney disease. This rare complication is assumed to be due to the hypodynamic circulatory state as a consequence of thyroid hormone deficiency. Paralytic ileus is even a rarer fatal manifestation of hypothyroidism and is thought to be due to autonomic neuropathy affecting the intestines that can be reversed by the thyroxine replacement.

Index Terms- Acute Kidney Injury, Hypothyroidism, Paralytic ileus, Hypokalemia.

I. INTRODUCTION

Hypothyroidism presenting with acute kidney injury is rare, with only few cases reported so far.[1,2] Hypothyroidism may induce de novo acute kidney injury (AKI) or may exacerbate ongoing chronic kidney disease[3] or contribute to the occurrence of AKI in the presence of other renal insults. AKI is life threatening and identification of possible contributory causes and early treatment is potentially lifesaving. Paralytic ileus is even a rarer fatal manifestation of hypothyroidism[4]. In this interesting case report a patient has been discussed who presented with severe hypothyroidism and both these complications.

II. CASE PRESENTATION

A 55 year old male patient, diagnosed with hypothyroidism four years ago, was admitted to our nephrology unit in a state of drowsiness and confusion. He was on thyroxine replacement therapy (150 µg daily) but was not compliant and stopped treatment for the last 3 months. He did not have any other comorbidities and the cause of hypothyroidism was unknown. In the month prior to time of admission, he had history of constipation, lethargy, and family members had noticed mental slowing, hoarse voice and changes in facial appearance. The patient was first admitted to a local hospital in a state of confusion and drowsiness with generalized body swelling, where oral thyroxine replacement was started. Patient was referred to our unit within 24 hour due to no improvement in clinical condition. On admission he had typical features of severe hypothyroidism such as coarse myxedematous skin, characteristic facial appearance, dry thin hairs and oedema. He was not hypothermic but was dehydrated. His heart rate was around 56/min, blood pressure was 156/90 mm Hg and there were no clinically detectable pericardial effusion. His respiratory system was clinically normal and abdomen was grossly distended with absent bowel sound, there was no organomegaly and free fluid. He was confused and drowsy (Glasgow coma scale; E-4, M-5, V-4) and had slow-relaxing reflexes.

The records shows that at initial presentation to the local hospital, his TSH (Thyroid Stimulating Hormone) level was 40.5 mIU/µl (Normal 0.4-4.0), Serum free thyroxine (FT4) level was 0.50 ng/dl (0.89-1.76) . One week after the initial presentation and while on thyroxine replacement, his TSH level had dropped to 30.2 mIU/µl, and the FT4 level was 0.61 ng/dl (0.89-1.76) but he was still severely hypothyroid. His serum creatinine had progressively risen from 1.38 mg% (on admission) to 5.11 mg%, with a concomitant drop in urine output (less than 20 ml per hour) over a span of 7th days. There were no active urinary sediments and USG abdomen showed normal kidney size with features of acute parenchymal disease together with bilateral fullness in the pelvicalyceal systems, rest was normal. The creatine phosphokinase (CPK-MM) and serum amylase level was normal. He was not on any nephrotoxic drugs. At the onset of renal impairment, the patient had hyponatraemia (114 meq/l) and was persistently hypokalemic (2.2-3.2 meq/l). Abdominal radiographs showed grossly dilated bowel loops with multiple air fluid levels. To rule out possibility of mechanical bowel obstruction, surgical opinion was consulted, and a surgical cause was thought to be unlikely, clinical features did not support peritonitis. So we kept first probable diagnosis of paralytic ileus and treatment started accordingly. Electrocardiogram was consistent with hypokalemia and there was no pericardial effusion on 2D - echocardiography. CT scan abdomen was not done because of the risk of contrast nephropathy aggravating existing AKI.

The patient was managed on the principles of thyroxine replacement therapy, steroid therapy and supportive therapy on the hypothesis that hypothyroidism and its sequalae were responsible for the clinical picture. Thyroxine 500 µg stat was

given and then replaced with levothyroxine (100 µg/day as standard maintenance dose) via nasogastric tube, intra-venous hydrocortisone 50 mg 6 hourly was started and dehydration was corrected with monitored administration of intravenous fluids with potassium replacement. The patient was nil orally except for the drug therapy. Intravenous levothyroxine is not available in our setting. With hydration and thyroxine replacement, the patient's renal functions improved (serum creatinine dropped to 2.34 mg %) together with increased urine output without renal replacement therapy. Hyponatremia also normalized without specific treatment. Bowel sounds returned on 6th day and oral feeding was started on 7th day with liquid feeds. The overall condition of the patient including the level of consciousness improved with thyroxine replacement. He became conscious, oriented and was subsequently mobilized within 15 days of hospital admission.

III. INVESTIGATIONS

Investigations	Before admission	At admission	1 week after admission	At discharge
TSH (normal 0.4-4.0)	40.5 mIU/µl	30.2 mIU/µl	22.4 mIU/µl	8.3 mIU/µl
S. Creatinine	-	1.38 mg%	5.11 mg%	2.34 mg%
S. Potassium	-	2.2 meq/l	3.2 meq/l	4.3 meq/l
S. Sodium	-	114 meq/l	136 meq/l	146 meq/l

IV. TREATMENT

He was on thyroxine replacement (150 µg daily) but stopped treatment 3 months back. On admission Thyroxine 500 µg stat was given and was then replaced with levothyroxine (100 µg/day as standard maintenance dose) via nasogastric tube, intra-venous hydrocortisone 50 mg 6 hourly was started and dehydration was corrected with monitored administration of intravenous fluids with potassium replacement. After two week of hospital stay patient was discharged in good condition and is in regular follow up.

V. DISCUSSION

There are several case reports of both acute renal failure[1,5] and paralytic ileus[6,7]occurring in untreated hypothyroidism, however both these complications appearing in the same patient has not been reported. The exact pathogenesis of acute kidney injury in hypothyroidism is still unclear and thought to be multi-factorial. However, the predominant mode of kidney injury is thought to be due to reduced plasma flow and glomerular filtration rate due to the hypodynamic circulation[2]. The hypodynamic circulatory state results in a pre-renal insufficiency and this may be aggravated by other multi-systemic effects of hypothyroidism such as reduced cardiac output, low volume status, hyponatremia with associated hemodynamic changes and increased peripheral resistance due to arterial wall stiffness[2]. However, this alone may not explain the extent of acute kidney injury. As per literature histological evidence from biopsy specimens show Primary glomerular and tubular dysfunction in hypothyroidism (thickening of glomerular and tubular basement membranes and inclusions in cell cytoplasm)[8]. These were reversible with thyroxine therapy. Rhabdomyolysis, another rare but known manifestation of hypothyroidism can also result in acute kidney injury but it is usually associated with another precipitating factor such as drugs or trauma[5]. Our patient however had no evidence of rhabdomyolysis. The available evidence suggests that renal impairment may start as quickly as two weeks in hypothyroid state and it recovers fully with thyroxine replacement[9]. The long term impact (if any) of hypothyroidism on renal function is unknown[5].

Paralytic ileus in hypothyroidism is assumed to be due to autonomic neuropathy affecting the extrinsic nerves of the colon[10]. There are only a few cases of this complication reported in literature with the first one being reported by Bastenie in 1946[7]. Bastenie hypothesized that myxoedematous material deposition in the muscle fibers of intestines interfered with their integration with autonomic ganglia. Later in 1969 and 1977, two case reports of death due to paralytic ileus with hypothyroidism were published[11,12,13]. In the report by Wells et al[11], the patient died after 20 days since presentation and the post mortem at that time gave an insight in to the possible pathogenesis in this unusual complication. Histological sections revealed gross abnormalities in extrinsic nerves innervating the intestines while some less prominent changes were also observed in intrinsic plexus. The authors suggest that the mechanism may be autonomic neuropathy similar to the peripheral neuropathy frequently observed in hypothyroidism. Surgical intervention for hypothyroidism induced paralytic ileus is not recommended as the neuropathy is reversible with thyroxine replacement. However, atonia may take time to reverse and the patients can succumb to complications of ileus[4].

In addition to paralytic ileus, patients may also present with urinary retention and this observation is also taken as supportive evidence for the autonomic neuropathy in hypothyroidism[4]. The fullness of the pelvicalyceal systems observed on ultrasound scan of our patient was possibly a result of this. We were unable to find any published data as to how the presence of paralytic ileus would have affected the absorption of thyroxine given via nasogastric tube; the non-availability of intravenous liothyronine was a definite, albeit unavoidable, shortcoming in our management.

In the chronology of events, it was noticed that the paralytic ileus precedes the acute kidney injury. The fluid sequestration in the bowels would have led to severe dehydration, hyponatraemia and hypokalaemia. In the patient's background and sequence of events, it is likely that hypothyroidism was the primary cause of paralytic ileus though subsequent hypokalaemia undoubtedly contributed to making it worse. It is unlikely that hypokalaemia was the primary cause of paralytic ileus since hypokalaemia developed later on. The acute kidney injury in this patient is unlikely to be due to rhabdomyolysis as there was no cause and supportive clinical or laboratory evidence. Instead, it is very likely that reduced renal plasma flow caused by hypothyroidism and the fluid sequestration within the intestines due to paralytic ileus in combination resulted in AKI. The paralytic ileus responded to potassium and thyroxine replacement and the concomitant vigorous fluid management would have improved the renal plasma flow. Both these therapeutic measures would have contributed to the rapid recovery of renal function. The relatively rapid recovery of renal function supports hypothyroidism related AKI rather than acute tubular necrosis due to dehydration.

In this patient, the root cause for paralytic ileus and acute kidney injury was the deficiency of thyroxine. The patient was managed on thyroxine replacement and supportive therapy leading to clinical improvement without surgery or invasive procedures, thus our diagnosis of paralytic ileus was correct.

VI. CONCLUSION

- It is important that clinicians are aware of the rare manifestations of hypothyroidism such as acute kidney injury and paralytic ileus.
- The easily reversible thyroxine deficiency may be missed when patients present with such complications unless there is an obvious past history.

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